Comp. Biochem. Physiol. Vol. 90B, No. 4, pp. 679–690, 1988
Printed in Great Britain

0305-0491/88 $3.00 + 0.00
© 1988 Pergamon Press plc

MINI-REVIEW

LIPID DYNAMICS IN FISH: ASPECTS OF ABSORPTION, TRANSPORTATION, DEPOSITION AND MOBILIZATION

MARK A. SHERIDAN
Department of Zoology, North Dakota State University, Fargo, ND 58105, USA (Tel.: 701-237-8110)

(*Received* 2 *September* 1987)

Abstract—1. Aspects of lipid metabolism, including absorption and depositional processes, appear quite different in fish as compared to homeothermic vertebrates.
2. Dietary lipids in fish are absorbed as fatty acids and as triacylglycerols aggregated into chylomicra particles.
3. Interorgan transport of lipids, like that of mammals, consists of an exogenous (dietary) loop and an endogenous loop.
4. Fish store lipids among several depot organs, including mesenteric membranes, liver and muscle.
5. Several fast-acting and slow-acting agents modulate depot lipid mobilization.
6. Mobilized lipids may be transported in the serum as free fatty acids bound to specific carrier proteins.

INTRODUCTION

Lipids serve a vast array of functions in the life histories of animals, ranging from, as Allen (1976) states, "the relatively mundane ... to (the) truly exotic ...". Entire symposia [i.e. *American Zoologist* 16 (4)] have been devoted to such topics. However, the most general and significant use of lipids is as energy reserves. The biochemical processing of lipids for energy metabolism by animals, including fish, can be described in eleven steps (Fig. 1). Aspects of lipid nutrition in fish, including digestion and utilization, have been extensively studied and thoroughly reviewed (Cowey and Sargent, 1977; Watanabe, 1982; Walton and Cowey, 1982; Greene and Selivonchick, 1987). This review will focus on the absorption, transport, deposition and mobilization of lipids in fish and discuss the hormonal mechanisms that serve to coordinate some of these processes.

ABSORPTION, TRANSPORT AND DEPOSITION

The lipid dynamics of mammals is well characterized. Dietary lipids, primarily triacylglycerols (TG), are hydrolyzed in the lumen of the gut by pancreatic lipases to free fatty acids (FFA) and 2-monoacylglycerols (2-MG) following emulsification into particles of 1000 nm in diameter with bile components (Masoro, 1968). Bile micelles comprised of bile salts, cholesterol and phospholipids take up the FFA and 2-MG forming mixed micelles of approximately 10 nm diameter. FFA and 2-MG diffuse from the mixed micelles into the mucosal cells of the jejunum. Within the mucosal cells, FFA greater than ten carbon atoms in length are activated on the smooth endoplasmic reticulum forming fatty acyl–CoA derivatives and are then re-esterified to 2-MG to reconstitute TG. Triacylglycerols are incorporated into chylomicra, large lipoprotein complexes of molecular weight up to 3×10^{10}, collect in the lymphatic system and ultimately enter the blood vascular system via the thoracic duct. Fatty acids of less than ten carbon atoms in length are sufficiently soluble in plasma to be transported as free solutes and leave the intestinal mucosa via the hepatic portal vein. Once in the liver these short-chain fatty acids can become incorporated into other lipoprotein classes, such as very low density lipoprotein (VLDL), and participate in the endogenous lipid transport system (Allen, 1976). Deposition of lipids into storage sites such as adipose tissue occurs when lipids in the chylomicra are hydrolyzed by lipoprotein lipase. Hydrolytic products, FFA and 2-MG, are absorbed and resynthesized into TG, the final storage product. Lipids are delivered to other peripheral tissues such as muscle on VLDL following lipoprotein lipase action. Aspects of lipid metabolism, including transport and deposition processes, appear quite different in fish as compared to homeothermic vertebrates.

Absorption

Early studies by Robinson and Mead (1973) and Kayama and Iijima, (1976) suggested that dietary lipids are absorbed and transported as FFA, a mechanism quite different from that defined for mammals. Robinson and Mead (1973) observed that rainbow trout fed ^{14}C-palmitate initially incorporated most of the radioactivity in the serum FFA fraction. Kayama and Iijima (1976) reported similar results in carp. Both studies, however, show that label in the FFA fraction declines dramatically after 2 hr and that 4 hr after feeding, label incorporation into the triacylglycerol fraction was increased (to levels as high as that noted for FFA 1 hr after feeding). The conclusion that FFA was the primary transport molecule was

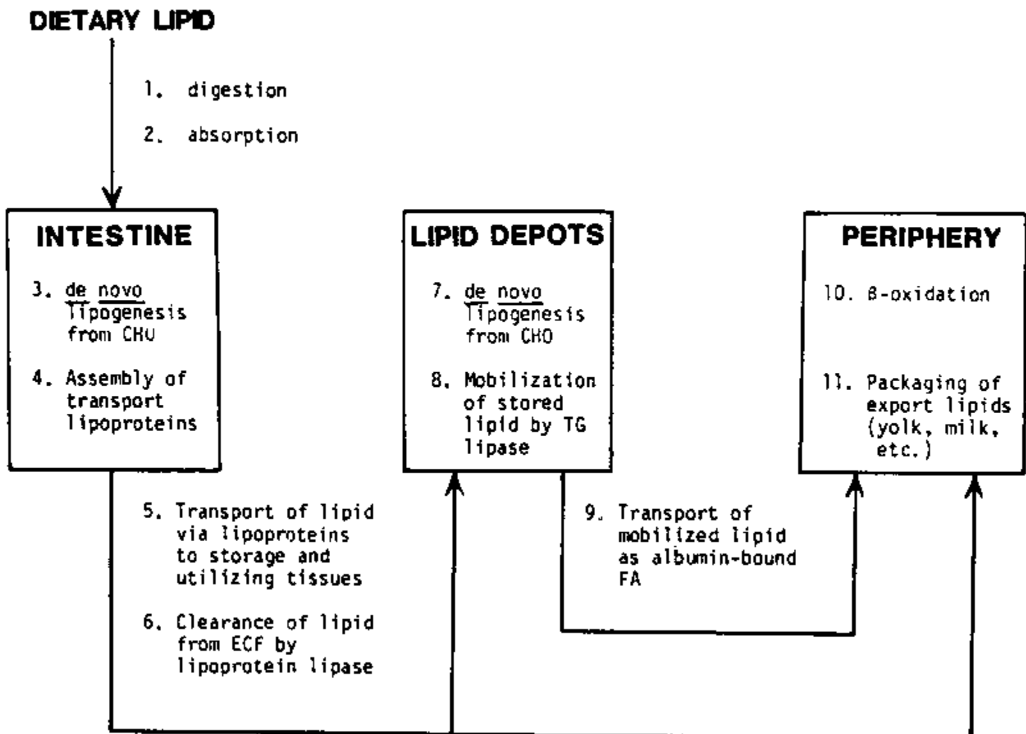

Fig. 1. Biochemical processes involved in lipid energy metabolism.

supported by the inability to observe large TG-rich chylomicra. More recently, however, ultrastructural evidence of Sire *et al.* (1981) has shown that re-esterification of the fatty acids does occur in the trout intestinal cell, followed by the emergence of a lipoprotein aggregate between 12–24 hr after investigation of a lipid load. Moreover, the size of the lipoprotein aggregate varies with the nature of the ingested meal. Particles range from 1000–1500 Å ("small chylomicra") in diameter after a standard meal, to <2600 Å diameter ("middle chylomicra") after a high fat meal, to <6500 Å diameter ("large chylomicra") after a meal containing a high proportion of ω3 polyunsaturated fatty acids (Sire and Vernier, 1981). Chylomicra particles have been isolated from trout serum (Rogie and Skinner, 1981; Sheridan *et al.*, 1985a), appearing as early as 2–4 hr after feeding (Fig. 2; Sheridan *et al.*, 1985a) and are

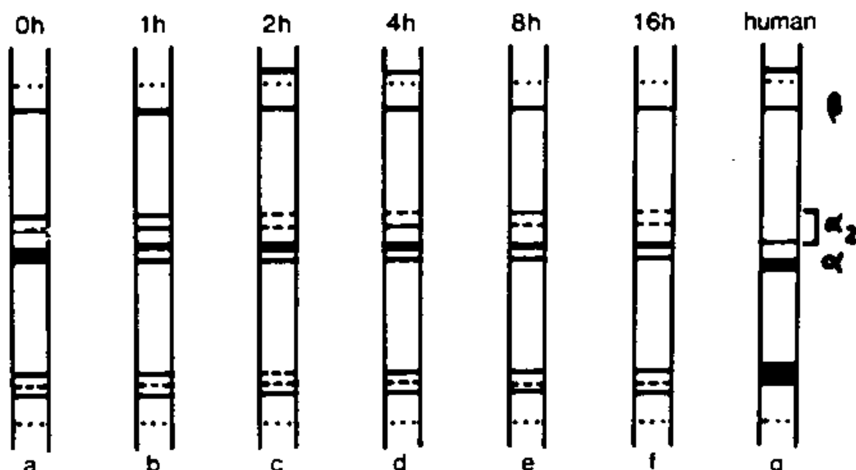

Fig. 2. Effects of lipid load on serum lipoprotein distribution in steelhead trout, *Salmo gairdnerii*. 10 μl of serum was incubated with 5 μl of Sudan Black B-ethylene glycol for 24 hr prior to electrophoresis. Dotted lines indicate boundary between stacking gel and separation gel, and the location of tracking dye; solid lines indicate major components; dashed lines indicate minor components. Number indicates hours after feeding; human serum reference 1 hr postprandial. (Reprinted with permission from Sheridan *et al.*, 1985a, Pergamon Press Journals).

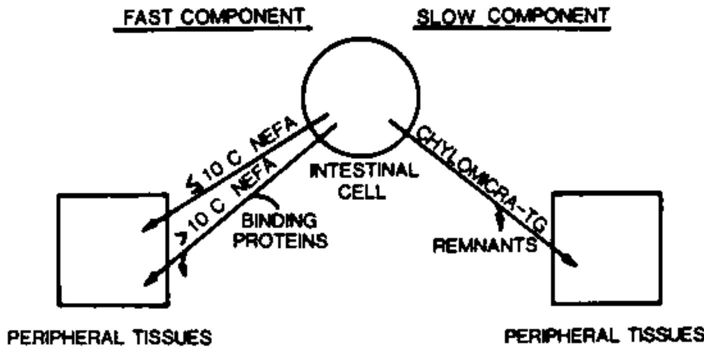

Fig. 3. Two-step lipid absorption model. Fast component is a fatty acid delivery system. Slow component represents a TG delivery system wherein TG are aggregated into chylomicra particles. NEFA, nonesterified fatty acids; TG, triacylglycerols. (Reprinted with permission from Sheridan *et al.*, 1985a, Pergamon Press.)

approximately 80% triacylglycerol (Sheridan *et al.*, 1985a). Rapid appearance (within 30 min) of FFA (Robinson and Mead, 1973; Kayama and Iijima, 1976) followed by the subsequent appearance of TG and chylomicra, lead Vernier and Sire (1983) and Sheridan *et al.* (1985a) to independently postulate a two-step lipid absorption model for fish (Fig. 3). The model consists of a fast component and a slow component. The fast component represents an FFA delivery system that consists of short-chain FFA that are soluble in plasma and longer-chain FFA that are presumably bound to carrier proteins. This component explains the radiolabel data of Robinson and Mead (1973) and of Kayama and Iijima (1976). The slower component represents a TG delivery system that consists of the aggregation, extrusion and transport of TG-rich particles, a mechanism similar to that used by mammals.

Transport

The interorgan transport of lipids in fish, like that of mammals, consists of two loops: an exogenous (dietary) loop and an endogenous loop (Fig. 4). The blood lipid compartment is a composite of newly-absorbed lipids, recently processed and repackaged lipids and lipids mobilized from storage sites. Figure 5 indicates the myriad of blood lipid components. As many as eight lipoprotein bands are observed in rainbow trout, these include proteins with α_1, α_2 and β mobility, as well as a fraction with chylomicra-like mobility (cf. Narayan *et al.*, 1966) and discrete proteins with albumin-like mobility. The chemical composition of several of the lipoproteins has been determined (Skinner and Rogie, 1978; Chapman *et al.*, 1978; Sheridan *et al.*, 1985a) and is summarized in Table 1. The proportion of lipids in any one fraction is dependent on a variety of factors, including nutritional state (cf. Sheridan *et al.*, 1985a), and developmental state, particularly stage of sexual maturity (Freemont and Marion, 1982).

The exogenous transport loop delivers lipid to active tissues and depot sites as chylomicra and fatty acids as discussed previously. Trout chylomicra are composed primarily of triacylglycerol, though minor amounts of cholesterol, cholesteryl esters and phospholipids are also present (Sheridan *et al.*, 1985a; Table 1). Protein components are associated with the chylomicra (Rogie and Skinner, 1985) and appear to

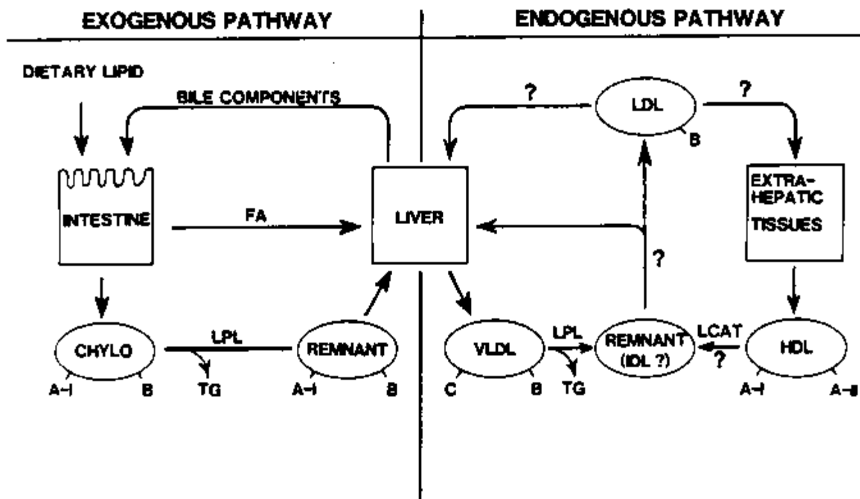

Fig. 4. Exogenous and endogenous lipid transport pathways in fish. (Adopted after mammalian model from Goldstein *et al.*, 1983.)

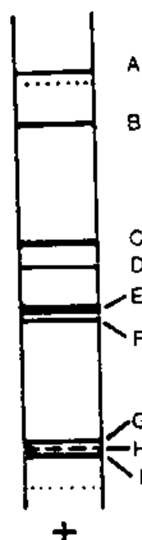

Fig. 5. Trout (*Salmo gairdneri*) serum lipoprotein components prestained with Sudan Black B as in Fig. 2 and separated by polyacrylamide gel electrophoresis (7.5% monomere concentration). The electrophoretic pattern depicted represents a typical 2 hr postprandial profile. Dotted lines indicate boundary between stacking gel and separation gel, and the location of tracking dye. A, chylomicra; B, very low density lipoprotein; C and D, low density lipoprotein components. C and D may also contain slow high density lipoprotein components. E and F, high density lipoprotein components, G–I, fatty acid binding proteins.

be similar to human apoproteins B and A-I. Dietary lipid delivered to the blood as fatty acids could be transported as free solute (chain lengths ≤10 carbons) or complexed to a protein carrier. Kayama and Iijima (1976), who observed an initial (1–3 hr) increase in labelled FFA in the plasma in carp force-fed FFA as ^{14}C-palmitic acid, showed that the majority of the label was associated with albumin-like lipoproteins. Lipid fed as TG (^{14}C-tripalmitin) did not result in an initial post-absorptive increase in label incorporated as FFA (Kayama and Iijima, 1976). Lipids are cleared from the plasma into first the liver and then into other storage/utilization sites (Robinson and Mead, 1973). Clearance is facilitated by lipoprotein lipases. Lipoprotein lipase activity has been observed in the plasma (Skinner and Youssef, 1982) and several tissues (Black *et al.*, 1982; Sheridan and Allen, 1984), including dark muscle, liver and adipose tissue of salmonids. The liver is the interface between the exogenous and endogenous transport loops.

The endogenous transport system delivers lipid to tissues and storage sites as VLDL, a large TG-rich lipoprotein (Table 1) which is secreted by the liver. The liver repackages dietary lipid and complexes it with lipid synthesized *de novo* and with protein. Much of the lipid is delivered in esterified form (Table 1); glyceryl esters predominate. Cholesterol, as is the case in mammals, is transported in both free and esterified form; however, a majority is as acyl esters. Trout VLDL possesses apoproteins similar to human apo B (Chapman *et al.*, 1978; Skinner and Rogie, 1978) and human apo C (Skinner and Rogie, 1978). Rogie and Skinner (1985) showed the synthesis of apo B and apo C in trout liver slices. In the capillaries and tissues of fish the VLDL triacyclglycerols are hydrolyzed. Whether the hydrolysis leads to the concomitant formation of intermediate density lipoprotein (IDL) as in mammals is not known (Fig. 4). The relatively cholesterol-rich low density lipoprotein (LDL) (Table 1) particle is present in fish plasma and is probably derived from VLDL, retaining apo B but having lost apo C during the conversion. Binding and cellular uptake of LDL in fish is yet to be demonstrated. Information is also lacking regarding the recycling of lipids in fish. Presumably, excess lipid (including cholesterol) is removed from extrahepatic tissue—perhaps by high density lipoprotein (HDL) in a manner similar to that seen in mammals. The apoprotein composition of trout HDL is similar to that of human HDL; trout HDL possessing apo A-I-like and apo A-II-like components (Skinner and Rogie, 1978; Chapman *et al.*, 1978).

When lipid stores in fish are mobilized, FFA are released into the plasma. The mobilized lipid generally remains unesterified and longer-(>10 carbon) chain FFA may be transported on carrier proteins. Fellows and Hird (1981) investigated FFA binding characteristics in fish and cyclostomes by thin-layer gel filtration. Serum of several teleosts and lamprey demonstrated FFA binding, whereas serum of several elasmobranchs did not exhibit FFA binding characteristics. Results from our laboratory (Sheridan and Allen, unpublished) indicated that the three proteins of trout plasma (Fig. 5) with albumin-like mobility all bound ^{14}C-palmitate as determined by liquid scintillation counting of polyacrylamide gel slices.

Deposition

Fish lipids appear to be stored among several depot organs rather than in one principal depot as is

Table 1. Lipid and protein composition of rainbow trout *Salmo gairdnerii* lipoproteins (%)

Component	Lipoprotein			
	Chylomicra	VLDL†	LDL†	HDL†
Triacylglycerol	74.6*	41.9/38.5	26.9/12.5	1.5/5.7
Cholesterol	2.2*	6.9/11.5	6.7/9.5	3.4/4.1
Cholesteryl ester	5.9*	15.1/26.7	15.6/27.9	7.7/20.1
Phospholipid	10.4*	26.5/16.1	27.1/14.9	26.5/27.9
Protein	6.7*	9.6/7.2	24.7/35.2	46.9/42.2
Major apoproteins	A-I, B‡	B/B, C	B/B	A-I, A-II/ A-I, A-II

*Data from Sheridan *et al.* (1985a).
†Data for VLDL, LDL, and HDL from Chapman *et al.* (1978) (before slash) and Skinner and Rogie (1978) (behind slash).
‡Data from Rogie and Skinner (1985).

Table 2. Lipid composition of various teleostean fish (mg/g fresh weight)

Species	Lipid class						Total
	PL	TG	CHOL	FA	CE	WE	
Trout							
Salmo gairdnerii[1]							
(Juvenile; FW parr)							
MF	6.2	529					527
DM	26	113	5.5				137
LM	20	46	3				62
L	22	17	1.2	0.06	0.72	1.23	46
S	7.5	11	2.3	1.5	1.6	2.2	25
S. gairdnerii[1]							
(Juvenile; FW smolt)							
MF	5.8	535					540
DM	39	10	0.2				47
LM	21	10	0.2				27
L	25	5	0.6	0.03	0.08	0.10	30
S	5.2	7	1.1	0.5	0.26	0.66	14
S. gairdnerii[2]							
(Adult FW resident)							
DM	10	34	5	2			51
LM	4	16	2	1.5			23
L[a]	9.6	2.9	6.2	4.4	3.1		26
Salmon							
Oncorhynchus kisutch[3]							
(Juvenile; FW parr)							
MF	19	590	17				625
DM	37	53	11				107
L	19.5	22	13	5	tr	tr	60
O. kisutch[3]							
(Juvenile; FW smolt)							
MF	17	525	18				580
DM	27	41	9				62
L	12	10	7.5	3	tr	tr	41
Whitefish[4,b]							
Coregomus albula							
(Adult)							
MF	10	←	—	20[c]	—	→	30
Roe	25.5	62.7	1.4	0.02	← 1.7[d] →		98
Tilapia[5]							
Oreochromis mossambicus							
LM	3.3		1.7[f]	17.8			66.5
L	31.6		15.6[f]	47.6			164.4
Gill	3.8		3.5[f]	8.5			76.9
Brain	80.4		25.5[f]	35			212.3
Bogue[6,b]							
Boops boops							
LM	5.3	12.1	0.3	tr			17.7
L	28.2	48.7	0.9	tr			77.8
head	12	144	2	tr			158
skin	8	169.3	1	tr			178
Haddock[7,b]							
Gadus aeglefinus							
LM	2.9	0.13	0.33	0.33	0.18	0.58[e]	5.50[g]
Cod[8,b]							
Gadus callarias							
LM	2.58	0.16	0.49	0.32	0.31	0.77[e]	6.04[g]

[1]Sheridan *et al.* (1983).
[2]Robinson and Mead (1973).
[3]Sheridan (1986).
[4]Kaitaranta (1980).
[5]Rao and Rao (1984).
[6]Kapoulas and Miniadis-Meimaroglou (1985).
[7]Garcia *et al.* (1956).
[8]Olley and Lovern (1954).
[a]Includes serum.
[b]Calculated from authors' original % composition data.
[c]Data for all neutral lipids combined.
[d]Data for CE and WE fractions combined.
[e]Data for WE and alcohols.
[f]Data for total cholesterol; includes both cholesterol and cholesteryl esters.
[g]Includes unidentified components and hydrocarbons.
Abbreviations: PL—phospholipid, TG—triacylglycerol, Chol—cholesterol, FA—fatty acid, CE—cholesteryl ester, WE—wax ester, MF—mesenteric fat, DM—dark muscle, LM—light muscle, L—liver, S—serum, FW—fresh water, tr—trace amounts.

the case with mammals. The primary storage molecule in higher bony fish (teleosts) is TG, however, glyceryl ether analogs (long-chain alcohols bound to glycerol via ether bonds) have been reported in certain tissues of some species (Kapoulas and Miniadis-Meimaroglou, 1985; Table 2). Some elasmobranchs use alkoxydiacylglycerols as depot lipids exclusively or in combination with other lipid classes, including TG (Lovern, 1962).

The major storage sites of fish are mesenteric fat, muscle and liver. Compositional data of various organs in several teleostean species are listed in Table 2. Braekken (1959) has suggested that the liver serves a major role in sluggish, bottom-dwelling fish, whereas skeletal muscle is important in more active groups. Such ecological correlates are difficult to support. For example, cod, which are deep-water feeders but hardly sluggish, have little lipid stored in the skeletal muscle and considerable amounts stored in the liver. Rainbow trout, which are active surface feeders, on the other hand, have substantial amounts of lipid stored in skeletal muscle and support Braekken's contention. Of the two skeletal muscle types, dark muscle appears to store more lipid than light muscle (Robinson and Mead, 1973; Sheridan *et al.*, 1983; Sheridan, 1986; Table 2). Robinson and Mead, (1973) showed that trout force-fed ^{14}C-palmitic acid incorporated radioactivity into dark muscle lipids 5-fold higher than into light muscle lipids.

Compositional data alone, however, do not indicate the dynamic nature of depot organs. Additional criteria, such as possession of lipid uptake and lipid breakdown systems reveal the physiological usefulness of the store. Lipoprotein lipase (the enzyme controlling lipid uptake in mammals) activity has been observed in the several depots (dark muscle, liver and mesenteric fat) of salmonids (Black *et al.*, 1982; Sheridan and Allen, 1984). Mobilization of lipids from depots is accomplished by activation of the lipolytic enzyme, TG lipase. Lipase activity has been observed in the liver, dark muscle and mesenteric fat of coho salmon, *O. kisutch* (Sheridan *et al.*, 1985b).

MOBILIZATION

The majority of the work on lipid mobilization has been done on mammalian and avian adipose tissue, the primary storage site for these animal groups. The hydrolysis of depot fat in the rat (Rizack, 1961; Vaughan *et al.*, 1964; Huttunen and Steinberg, 1971; Arnaud and Boyer, 1974; Pittman *et al.*, 1975; Fredrikson *et al.*, 1981) and the chicken (Khoo *et al.*, 1976) is catalyzed by "hormone-sensitive" triacylglycerol (TG) lipase, diacylglycerol hydrolase, monoacylglycerol hydrolase and cholesteryl ester hydrolase. The lipolytic action of these enzymes results in the mobilization of FFA (Fredrickson and Gordon, 1958; Vaughan and Steinberg, 1965) and the subsequent transport of these acyl acids to peripheral tissues (Allen, 1976). Therefore, experimental criteria for lipid mobilization are (1) depletion of tissue total lipid, (2) depletion of tissue triacylglycerol, (3) enhanced lipase activity, (4) increased *in vitro* FFA release, and (5) elevated plasma FFA. The hormone-sensitivity of the mammalian TG lipase (EC 3.1.1.2) in adipose tissue has been well-demonstrated (for review, see Fain, 1980). The charactersitics of the various adipose enzyme preparations are rather uniform with regard to pH optima. Optimum activity occurs at about 7.5 (Rizack, 1961; Vaughan *et al.*, 1964; Huttunen and Steinberg, 1971; Arnaud and Boyer, 1974; Khoo *et al.*, 1976; Fredrikson *et al.*, 1981) for the cytosolic enzyme, which is separable from the hydrolase activity occurring at pH 5.0 that appears to be associated with membrane elements (Mahadervan and Tappel, 1968; Guder *et al.*, 1969). The picture of mammalian hepatic lipid utilization appears quite different. Lipolytic activity in rat liver homogenates has been observed in lysosomal, microsomal, cytosolic and plasma membrane cell fractions, though Debeer *et al.* (1982) suggest that the liver contains two types of lipases: one intracellular acid lipase (pH optimum of 4–5) located in the lysosomes and one heparin-releasable enzyme with alkaline pH optimum (8–9.5) which is mainly associated with the exterior face of plasma membranes of non-parenchymal cells. The alkaline lipase is probably the lipoprotein lipase controlling lipid uptake mentioned previously.

Substantially less information is known about lipid mobilization in poikilotherms. Lipolytic activity toward long-chain triacylglycerols has been observed in salmonid dark muscle (Bilinski and Lau, 1969; Sheridan *et al.*, 1985b) liver (Sheridan *et al.*, 1985b) and adipose tissue (Sheridan and Allen, 1984). Maximum enzyme activity in the various tissues ranges from pH 7.3 to 7.5. Lysosomal acid lipase activity has been observed in trout dark muscle (Bilinski *et al.*, 1971) and adipose tissue (Sheridan and Allen, 1984); although complete purification procedures and characterization have only been reported for trout adipose tissue (Sheridan and Allen, 1984). The neutral lipase isolated from trout adipose tissue has a molecular weight of about 48,000. In the few homeothermic species in which purified lipase preparations have been obtained, a wide range of molecular weight proteins have been identified. In the rat, an $M_r = 85,000$ protein has been identified by a number of investigators (Belfrage *et al.*, 1977; Khoo *et al.*, 1980; Fredrikson, 1981). Berglund *et al.* (1980) isolated a $M_r = 42,000$ protein from chicken adipose tissue that was capable of being phosphorylated when incubated with cAMP-dependent protein kinase and ATP–Mg. In sum, it appears as though neutral lipase is important in controlling lipid mobilization. The physiological significance of the acid lipase is less clear. The physiological function of lysosomes, where acid lipase occurs, is thought to consist of intracellular digestion of cell constituents. Bilinski *et al.* (1971) point out that trout lateral line muscle is very active and suggests that the lysosomal lipase may serve to mobilize intracellular lipid stores for internal utilization. This action would be considerably different from the role of neutral lipase in adipose tissue where mobilization of lipids occurs so that the stores can be utilized by peripheral tissues. Certainly the function of hepatic lipases, both acid lipases as found in mammals, and neutral lipases as found in fish, need to be examined more closely.

Hormonal control of lipid mobilization

The hormonal regulation of lipid mobilization from mammalian adipose tissue has been well studied. Catecholamines, thyroid hormones, glucocorticoids, growth hormone and prolactin are well-known adipokinetic agents (see reviews: Vaughan, 1961; Jeanrenaud, 1961; Meisner and Carter, 1977; Fain, 1980). In mammalian adipocyte preparations, norepinephrine (NE) is more potent than epinephrine in stimulating lipolysis and appears to activate TG lipase via $beta_1$ adrenoreceptors (Fain and Garcia-Sainz, 1983). Triiodothyronine (T_3) increases metabolic rate and plasma FFA levels without causing alterations in plasma glucose (Rich *et al.*, 1959). Propylthiouracil-induced hypothyroidism depresses epinephrine-stimulated lipolysis in rats (Debons and Schwartz, 1961), suggesting a modulative role of thyroxin in lipid mobilization, perhaps mediated by thyroid hormone effects on cAMP accumulation (Malbon *et al.*, 1978). The effects of glucocorticoids in mammals appears to be mediated through cAMP-independent pathways (Fain, 1980). These effects on lipid mobilization involve both stimulation of lipolysis (TG hydrolysis) and inhibition of FFA re-esterification via inhibition of glucose transport into fat cells (Fain, 1980). Growth hormone-stimulated lipolysis in rat adipocytes occurs after a one hour lag period and differs from that of fast-acting agents in its sensitivity to inhibitors of RNA and protein synthesis and probably operates through DNA-dependent RNA synthesis (Fain *et al.*, 1965). However, growth hormone administration does result in cAMP accumulation, though the growth hormone-stimulated cAMP accumulation can be blocked by puromycin or cyclohexamide (Fain *et al.*, 1971). Taken together, the accumulated evidence suggests that lipolysis in mammalian fat cells is stimulated by multiple factors, some operating through more than one mechanism of action.

In fish, the metabolic effects of hormones are less well studied and investigations employing species-specific hormones for physiological studies on fish are rare (see Plisetskaya, 1980; Plisetskaya *et al.*, 1983). Therefore, the majority of the accumulated evidence has come from *in vivo* studies, providing little insight as to mechanism of action. Agents known to enhance lipid mobilization in fish are listed in Table 3; demonstrated experimental criteria are noted for each. Evidence on catecholamine action in fish is conflicting. Farkas (1967a,b, 1968, 1969a,b) has consistently failed to stimulate lipolysis with catecholamines. *In vivo* administration of NE to carp leads to a slight decline in plasma FFA; *in vivo* or *in vitro* exposure to NE tends to decrease FFA release from carp adipose tissue (1969a). Farkas (1969a) suggests that these effects on lipid metabolism are mediated through effects on carbohydrate metabolism (hyperglycemic) and result in the re-esterification of FFA. In scorpionfish, lamprey (Leibson *et al.*, 1968; Plisetskaya and Mazina, 1969) and eel (Larsson, 1973), however, catecholamines cause an increase in plasma FFA. The bases of the conflicting evidence for catecholamine action may lie in the distribution of lipids among storage sites and in the particular tissue under study. The pattern which emerges from the accumulated evidence, including results from our laboratory, suggest that adipose tissue fails to respond to catecholamine treatment (Farkas, 1969a; Murat *et al.*, 1985; Sheridan, unpublished observations), whereas liver slices incubated *in vitro* respond rapidly to NE treatment (Sheridan, 1983, 1987). NE-stimulated decreases in 1-^{14}C-acetate incorporation into hepatic lipids of the nurse shark (Lipshaw *et al.*, 1972) support a lipolytic effect of catecholamines on fish liver. Catecholamine action on salmon liver appears to depend on the nature of the agent. NE induces rapid lipolysis, whereas epinephrine failed to have a significant lipolytic effect (Sheridan, 1987). Activation of lipolysis in salmon liver proceeds from the activation of β-receptors and results in the stimulation of TG lipase.

A host of peptides have been reported to have a lipolytic effect in fish. Adrenocorticotropic hormone (ACTH) (100 IU/kg) increased plasma FFA in goldfish 6–24 hr after administration (Minick and Chavin, 1970). However, ACTH had no effect on plasma FFA in carp (Farkas, 1967a) and rainbow trout (Takashima *et al.*, 1972). Farkas investigated the *in vitro* effect of ACTH in carp (1967a), pike, perch and bream (1969a) by analysing tissue FA content and found that ACTH had no effect on adipose tissue fatty acid content. The significance of these *in vitro* results, however, is unclear since mobilization of stored triacylglycerol results in release of fatty acid from cells (into media). The tissue fatty acid pool may indeed be quite slight with the most potent adipokinetic agent when removal by albumin is rapid. In cultures devoid of albumin, as were those of Farkas, significant re-esterification of FFA may occur and mask any lipolytic effects.

The role of glucagon on lipid mobilization in fish is ill-defined. Mammalian glucagon has no effect on plasma FFA in toadfish (Tashima and Cahill, 1968), eel (Larsson and Lewander, 1972; Chan and Woo, 1978) and lamprey (Plisetskaya and Mazina, 1969). Intra-arterial administration of mammalian glucagon into pike, however, resulted in elevated plasma FFA in serial samples 6–9 hr after injection (Ince and Thorpe, 1975). *In vivo* administration of mammalian glucagon in coho salmon stimulated liver triacylglycerol lipase activity in a dose-dependent manner; minimal effective dose equals 1 μg/mL after 3 hr after injection (Sheridan, unpublished). *In vivo* administration of salmon glucagon similarly stimulates liver lipase activity in salmon (Sheridan, unpublished).

Somatostatin-14 (SRIF), well-known for its inhibitory actions on the release of various pituitary and pancreatic hormones, is the most investigated form of somatostatins. Other *N*-terminally extended forms containing 22, 25 or 28 amino acids have been characterized (cf. Plisetskaya *et al.*, 1983). Intraperitoneal injection of salmon somatostatin-25 (sSS-25) into juvenile coho salmon results in a dose-dependent increase in plasma FFA and a transient hyperglycemia (Sheridan *et al.*, 1987). These changes were accompanied by an initial (1 hr) somatostatin-induced suppression of insulin titers, and by enhanced lipase activity in the liver and adipose tissue. The direct effects of SRIF on lipid mobilization in salmon have also been investigated. SRIF stimulated an almost instantaneous dose-dependent release of

Table 3. Agents that increase lipid mobilization in fish

Agent	Lag period	Features
Epinephrine	? *in vivo* effect in 1 hr	increases plasma FA[1]
Norepinephrine	seconds	increases FA release[2] increases lipase activity[2]
ACTH	? *in vivo* effect in 6–24 hr	increases plasma FA[3]
mGlucagon	? *in vivo* effect in 3 hr	increases plasma FA[4] increases lipase activity[5]
sGlucagon	? *in vivo* effect in 3 hr	increases lipase activity[5]
Somatostatin-14	seconds	increases FA release[6] increases lipase activity[6]
sSomatostatin-25	? *in vivo* effect in 3 hr	increases plasma FA[7] increases lipase activity[7]
Urotensin II*	seconds	increases FA release[6] increases lipase activity[6]
Arginine vasotocin	? *in vivo* effect in 30 min	increases plasma FA[8,9]
Thyroxin	? *in vivo* effect in 24 hr	increases plasma FA[10] decreases stored TG[11-14] increases lipase activity[14]
Cortisol	? *in vivo* effect in 10 days	increases plasma FA[15] decreases stored TG[14] increases lipase activity[14]
Diethylstilbestrol	? *in vivo* effect in 8 days	increases plasma total lipids[16]
bGrowth hormone	? *in vivo* effect in 2 weeks	increases lipase activity[14]
oGrowth hormone	? *in vivo* effect in 3–8 hr	increases plasma FA[3]
oProlactin	? *in vivo* effect in 3–8 hr	increases plasma FA[3,17] decreases stored TG[14,18] increases lipase activity[14]
dbcAMP	seconds	increases FA release[6] increases lipase activity[6]
3-isobutyl-1-methylxanthine (IBMX)	seconds	increases FA release[6] increases lipase activity[6]

[1]Larsson (1973).
[2]Sheridan (1987).
[3]Minick and Chavin (1970).
[4]Ince and Thorpe (1975).
[5]Sheridan (unpublished).
[6]Sheridan and Bern (1986).
[7]Sheridan *et al.* (1987).
[8]McKeown *et al.* (1976).
[9]John *et al.* (1977).
[10]Murat and Serfaty (1970).
[11]Barrington *et al.* (1961).
[12]Narayansingh and Eales (1975).
[13]Singh (1979).
[14]Sheridan (1986).
[15]Butler (1973).
[16]Takashima *et al.* (1972).
[17]Leatherland *et al.* (1974).
[18]Meier (1972).
Species designations: m—mammalian (mixture of bovine and porcine), s—salmon, b—bovine, o—ovine, *—synthetic *Gillichthys*.
Abbreviations: TG—triacylglycerols, FA—fatty acid(s).

FFA from coho salmon liver incubated *in vitro* (Sheridan and Bern, 1986).

Urotensin-II (UII) is a dodecapeptide secreted by the caudal neurosecretory system of teleost fish and has partial analogy and partial homology to SRIF. *In vivo* administration of UII, like sSS-25, stimulated a dose-dependent increase in plasma FFA of coho salmon (Sheridan *et al.*, 1987). Injection of UII also resulted in enhanced depot (liver and adipose tissue) lipase activity. UII also stimulates fatty acid release from, and lipase activity in, coho salmon liver incubated *in vitro* (Sheridan and Bern, 1986).

Arginine vasotocin (AVT), a neurohypophysial hormone, is found in most classes of non-mammalian vertebrates. AVT injected into either juvenile coho salmon (15 mU/fish; McKeown *et al.*, 1976) or lam-

prey (1000 mU/fish; John *et al.*, 1977) significantly elevated plasma FFA 30 and 90 min after injection, respectively. AVT injected at a dose of 150 mU/fish decreased plasma FFA levels in salmon within 30 min (McKeown *et al.*, 1976).

Thyroid hormones tend to reduce stored body fat (Barrington *et al.*, 1961; Narayansingh and Eales, 1975 and Singh, 1979) in fish. Radiothyroidectomy causes an increase in mesenteric fat content in rainbow trout (Norris, 1969). Injection of thyroxin (T_4) into carp results in elevated plasma FFA 24 hr after injection (Murat and Serfaty, 1970). Takashima *et al.* (1972) reported, however, that mammalian thyroid powder (500 mg/fish) decreased plasma FFA in rainbow trout by half. Chronic exposure of juvenile coho salmon parr to T_4 for 14 days results in lipid mobilization from several storage sites: mesenteric fat, dark muscle and liver (Sheridan, 1986). Although the mobilization scheme varies among tissues, generally, total lipids were depleted primarily from the triacylglycerol fraction and the depletion was accompanied by enhanced lipase activity. Coho salmon smolts appeared refractory to T_4 treatment (Sheridan, 1986).

Cortisol injections increase plasma FA in the American eel, *Anguilla rostrata* (Butler, 1973), but not in the European eel, *A. anguilla* (Larsson and Fange, 1977). Implantation of juvenile coho salmon parr with cortisol results in lipid depletion, primarily as triacylglycerols, accompanied by elevated lipase activity in the liver, dark muscle and mesenteric fat (Sheridan, 1986). Cortisol treatment failed to elicit lipid mobilization in coho salmon smolts. The influence of sex steroids on plasma lipid levels was investigated by Takashima *et al.* (1972). Diethylstilbestrol (DES; 2000 μg/fish) injected into adult rainbow trout, *Salmo gairdnerii*, increased the plasma lipid content from 2.8 g/dl to 4.0 g/dl. Methyl testosterone, however, had no effect on plasma lipids of trout (Takashima *et al.*, 1972).

Growth hormone (GH) has varied and conflicting effects upon lipid metabolism in fish and in lower vertebrates generally. Mammalian GH causes increased lipid storage in lizards (Licht and Hoyer, 1968), has little effect on lipid storage in turtles (Nichols, 1973), and tends to decrease lipid storage in salmon (Clarke, 1976). Length of GH exposure is a complication which arises when assessing GH action. Long exposure to GH results in lipolytic effects, whereas short exposure periods usually result in lipogenic (insulin-like) action (Goodman and Schwartz, 1974). With long exposure, ovine GH has the effect of increasing plasma FFA in goldfish (Minick and Chavin, 1970) and bovine GH enhances lipase activity in juvenile coho salmon parr depots (Sheridan, 1986). The stage of development also appears to affect GH action. Juvenile coho salmon smolts, further along in sea water preadaptive development than parr, are refractory to 14 days of GH exposure with regard to lipid mobilization (Sheridan, 1986). Smolt refractoriness is further supported by the report of McKeown *et al.* (1975) that GH exposure had no effect on plasma FFA levels in smoltified kokanee salmon (*O. nerka*).

Prolactin (PRL), like GH, undoubtedly plays a role in the lipid metabolism of fish, but the actions of PRL are often subject to rhythmicity and interactions with other factors (cf. Meier, 1972). Ovine PRL has been reported to increase plasma FFA in goldfish (Minick and Chavin, 1970). Leatherland *et al.* (1974) observed a circadian rhythmicity in kokanee salmon plasma PRL was followed by a rhythmicity in plasma FFA. The metabolic response to PRL appears to vary with time of injection. Prolactin injections given early in the day stimulate lipolysis, whereas injections given later in the day favor fattening (deVlaming and Sage, 1972). Results from our laboratory contribute further to the concept of rhythmicity of PRL action on lipid metabolism. *In vivo* implantation of PRL into juvenile coho salmon parr for 14 days strongly stimulates lipid mobilization (Sheridan, 1986). The mobilization pattern generally consisted of reduced triacylglycerol content and enhanced lipase activity in the several depots (liver, dark muscle and mesenteric fat). In salmon smolts, however, PRL treatment, like GH, thyroid and cortisol, had little effect on lipid mobilization. Such developmental variation in PRL effects on lipid mobilization were also observed in kokanee salmon where PRL injection into smolts failed to increase plasma FFA (McKeown *et al.*, 1975). The effects of PRL on lipid metabolism also appears to involve a synergism with other hormones, particularly corticosteroids. Prolactin injected in killifish (*Fundulus grandis*) 18 hr after a previous injection of cortisol promoted fat deposition, whereas PRL injected only 6 hr after steroid pretreatment promoted lipid mobilization.

Lipolysis in liver slices is also stimulated by cAMP, suggesting an adenylate cyclase mechanism of action (though there may be other mechanisms operative also). Exogenous cAMP, administered to incubation medium as dibutyryl cAMP (dbcAMP), stimulates FFA release from coho salmon liver slices (Sheridan and Bern, 1986). Elevated levels of intracellular cAMP, achieved by inhibition of phosphodiesterase with 3-isobutyl-1-methylxanthine (IBMX), similarly stimulated lipid mobilization in salmon liver (Sheridan and Bern, 1986).

SUMMARY AND CONCLUSION

In fish, lipids are absorbed as either fatty acids or as triacylglycerols aggregated into chylomicra particles. The interorgan transport of lipid in fish is generally similar to that of mammals. Fish possess both an exogenous and an endogenous transport system. The lipoprotein complexes of the endogenous transport system resemble those of other animals; certain apoprotein constituents are similar to human apoproteins. The interconversion of lipoprotein in fish is not well-defined as is the mechanism of LDL-cholesterol uptake. Lipids in fish are stored in several depot organs (liver, muscle, mesenteric fat) primarily as triacylglycerol. Mobilization of lipids proceeds from the activation of lipolytic enzyme activity (triacylglycerol lipase) and results in the hydrolysis of stored TG and subsequent release of FA. Fatty acids are carried in the plasma of fish by one, or perhaps more, albumin-like binding proteins. Lipolytic enzyme activity is hormonally modulated. A variety of slow acting (thyroxin, cortisol, growth hormone, prolactin) and fast-acting (epinephrine, nor-

epinephrine, somatostatin) agents have been shown to stimulate lipase activity; perhaps indicating multiple mechanisms of hormone action.

REFERENCES

Allen W. V. (1976) Biochemical aspects of lipid storage and utilization in animals. *Am. Zool.* **16**, 631–647.

Arnaud J. and Boyer J. (1974) Identification of an acyl-cholesterol lipase activity in human adipose tissue. *Biochim. biophys. Acta* **337**, 165–168.

Barrington E. J. W., Barron N. and Piggins D. J. (1961) The influence of thyroid powder and thyroxine upon the growth of rainbow trout (*Salmo gairdnerii*). *Gen. comp. Endocr.* **1**, 170–178.

Belfrage P., Jergil B., Stralfors P. and Tornqvist H. (1977) Hormone-sensitive lipase of rat adipose tissue: Identification and some properties of the enzyme protein. *FEBS Lett.* **75**, 259–264.

Berglund L., Khoo J. C., Jensen D. and Steinberg D. (1980) Resolution of hormone-sensitive lipase triglyceride/diglyceride lipase from monoglyceride lipase of chicken adipose tissue. *J. biol. Chem.* **255**, 5420–5428.

Bilinski E. and Lau Y. C. (1969) Lipolytic activity toward long-chain triglycerides in lateral line muscle of rainbow trout (*Salmo gairdnerii*). *J. Fish. Res. Bd Can.* **26**, 1837–1866.

Bilinski E., Jonas R. E. and Lau Y. C. (1971) Lysosomal triglyceride lipase from lateral line tissue of rainbow trout (*Salmo gairdnerii*). *J. Fish. Res. Bd Can.* **28**, 1015–1018.

Black D., Youssef A. M. and Skinner E. R. (1983) The mechanism of lipid uptake by tissues in rainbow trout. *Biochem. Soc. Trans.* **11**, 93–94.

Braekken O. R. (1959) A comparative study of vitamins in the trunk muscles of fishes. *Rep. Technol. Res. Norw. Fish. Ind.* **3**(8), 1–51.

Butler D. C. (1973) Structure and function of the adrenal gland of fishes. *Am. Zool.* **13**, 839–879.

Chan D. K. O. and Woo N. Y. S. (1978) Effect of glucagon on the metabolism of the eel, *Anguilla japonica*. *Gen. comp. Endocr.* **35**, 216–225.

Chapman M. J., Goldstein S., Mills G. L. and Leger C. (1978) Distribution and characterization of serum lipoproteins and their apolipoproteins in the rainbow trout (*Salmo gairdnerii*). *Biochemistry* **7**, 4455–4464.

Clarke W. C. (1976) Effect of prolactin and growth hormone on growth, lipid content and seawater adaptation in juvenile sockeye salmon. *Reg. Conf. Comp. Endocrinol., Corvallis, OR.* (abstract).

Cowey C. B. and Sargent J. R. (1977) Lipid nutrition in fish. *Comp. Biochem. Physiol.* **57B**, 269–274.

Debeer L. J., Beynen A. C., Mannaerts G. P. and Geelen M. J. H. (1982) Lipolysis of hepatic triacylglycerol stores. *FEBS Lett.* **140**, 159–164.

Debons A. F. and Schwartz I. L. (1961) Dependence of the lipolytic action of epinephrine *in vitro* upon thyroid hormone. *J. Lipid Res.* **2**, 86–89.

Fain J. N., Kovacev V. P. and Scow R. O. (1965) Effect of growth hormone and dexamethasone on lipolysis and metabolism in isolated fat cells of the rat. *J. biol. Chem.* **240**, 3522–3529.

Fain J. N., Dodd A. and Novak L. (1971) Relationship of protein synthesis and cyclic AMP to lipolytic action of growth hormone and glucacorticoids. *Metabolism* **20**, 109–118.

Fain J. N. (1980) Hormonal regulation of lipid mobilization from adipose tissue. In *Biochemical Actions of Hormones* (Edited by Litwack G.). Vol. III, pp. 120–204, Academic Press, New York.

Fain J. N. and Garcia-Sainz J. A. (1983) Adrenergic regulation of adipocyte metabolism. *J. Lipid Res.* **24**, 945–966.

Farkas T. (1967a) The effect of catecholamines and adrenocorticotropic hormone on blood and adipocyte tissue FFA levels in the fish *Cyprinus carpio* L. *Prog. Biochem. Pharmac.* **3**, 314–319.

Farkas T. (1967b) Examinations on the fat metabolism in freshwater fishes. The sympathetic nervous system and the mobilization of fatty acids. *Ann. Inst. Biol. Hung. Acad. Sci.* (*Tihany*) **34**, 129–138.

Farkas T. (1968) The effects of some adrenergic blocking agents on the mobilization of fatty acids in lower vertebrates. *Ann. Inst. Biol. Hung. Acad. Sci.* (*Tihany*) **35**, 127–135.

Farkas T. (1969a) Studies on the mobilization of fats in lower vertebrates. *Acta Biochim. Biophys. Acad. Sci.* (*Hungary*) **4**, 237–249.

Farkas T. (1969b) Effects of agents modifying the level of cyclic 3′-5′-adenosine monophosphate in adipose tissue on mobilization of fats in fish and frogs. *Ann. Inst. Biol. Hung. Acad. Sci.* (*Tihany*) **36**, 163–171.

Fellows F. C. I. and Hird F. J. R. (1981) Fatty acid binding proteins in the serum of various animals. *Comp. Biochem. Physiol.* **68B**, 83–87.

Fredrickson D. S. and Gordon R. S. (1958) Transport of fatty acids. *Physiol. Rev.* **8**, 585–630.

Fredrikson G., Stralfors P., Nilsson N. O. and Belfrage P. (1981) Hormone-sensitive lipase of rat adipose tissue: purification and properties. *J. biol. Chem.* **256**, 6311–6320.

Freemont L. and Marion D. (1982) A comparison of the lipoprotein profiles in male trout (*Salmo gairdnerii*) before maturity and during spermiation. *Comp. Biochem. Physiol.* **73B**, 849–855.

Garcia M. D., Lovern J. A. and Olley J. (1956) The lipids of fish—6. The lipids of cod flesh. *Biochem. J.* **62**, 99–107.

Goldstein J. L., Kita T. and Brown M. S. (1983) Defective lipoprotein receptors and atherosclerosis: lessons from an animal counterpart of Familial hypercholesterolemia. *New Engl. J. Med.* **309**, 288–296.

Goodman H. M. and Schwartz J. (1974) Growth hormone and lipid metabolism. In *Handbook of Physiology* (Edited by Greep R. O. and Astwood E. B.), Sect. 7, Vol. 4, Part 2, pp. 211–231. Williams & Wilkins, Baltimore.

Guder W., Weiss L. and Weiland O. (1969) Triglyceride breakdown in rat liver: the demonstration of three different lipases. *Biochim. biophys. Acta* **187**, 173–185.

Greene D. H. S. and Selivonchick D. P. (1987) Lipid metabolism in fish. *Prog. Lipid Res.* **26**, 53–85.

Huttunen J. K. and Steinberg D. (1971) Activation and phosphorylation of purified adipose tissue hormone-sensitive lipase by cyclic AMP-dependent protein kinase. *Biochim. biophys. Acta* **239**, 411–427.

Ince B. W. and Thorpe A. (1975) Hormonal and metabolite effects on plasma free fatty acids in the northern pike, *Esox lucius* L. *Gen. comp. Endocr.* **27**, 144–152.

Jeanrenaud B. (1961) Dynamic aspects of adipose tissue metabolism: a review. *Metabolism* **10**, 535–581.

John T. M., Thomas E., George J. C. and Beamish F. W. H. (1977) Effect of vasotocin on plasma free fatty acid level in the migrating anadromous sea lamprey. *Arch. Int. Physiol. Biochim.* **85**, 865–870.

Kaitaranta J. K. (1980) Lipids and fatty acids of a whitefish (*Coregonus albula*) flesh and roe. *J. Sci. Fd Agric.* **31**, 1303–1308.

Kapoulas V. M. and Miniadis-Meimaroglou S. (1985) Composition and distribution of lipids in tissues of Bogue (*Boops boops*). *Z. Naturforsch.* **40C**, 562–565.

Kayama M. and Iijima N. (1976) Studies on lipid transport mechanisms in the fish. *Bull. Jap. Soc. Sci. Fish.* **42**, 987–996.

Khoo J. C., Steinberg D., Huang J. J. and Vagelos D. R. (1976) Triglyceride, diglyceride, monoglyceride, and cholesterol ester hydrolases in chicken adipose tissue activated by adenosine 3′:5′-monophosphate-dependent protein kinase. *J. biol. Chem.* **251**, 2882–2890.

Khoo J. C., Berglund L., Jensen D. and Steinberg D. (1980) Hormone-sensitive lipase of rat adipose tissue. Cor-

relation of activity with a protein of molecular weight 84,000. *Biochim. biophys. Acta* **619**, 440.

Larsson A. and Lewander K. (1972) Effects of glucagon administration to eels (*Anguilla anguilla* L.). *Comp. Biochem. Physiol.* **43A**, 831–836.

Larsson A. C. (1973) Metabolic effects of epinephrine and norepinephrine in the eel, *Anguilla anguilla* L. *Gen. comp. Endocr.* **20**, 155–167.

Larsson A. and Fange R. (1977) Cholesterol and free fatty acids (FFA) in the blood of marine fish. *Comp. Biochem. Physiol.* **57B**, 191–196.

Leatherland J. F., McKeown B. A. and John T. M. (1974) Circadian rhythm of plasma prolactin, growth hormone, glucose and free fatty acids in juvenile kokanee Salmon, *Oncorhynchus nerka*. *Comp. Biochem. Physiol.* **47**, 821–828.

Leibson L. G., Plisetskaya E. and Mazina T. I. (1968) The NEFA content in the blood of cyclostomata and fishes and the effect of epinephrine and insulin. *J. Evol. Biochem. Physiol.* **4**, 121–127.

Licht P. and Hoyer H. (1968) Somatotropic effects of exogenous prolactin and growth hormone in juvenile lizards (*Lacertas sicula* S.). *Gen. comp. Endocr.* **11**, 338–346.

Lipshaw L. A., Patent G. H. and Foa P. P. (1972) Effects of epinephrine and norepinephrine on the hepatic lipids of the nurse shark, *Ginglymostoma cirratum*. *Horm. Metab. Res.* **4**, 34–38.

Lovern J. A. (1962) The lipids of fish and changes occurring in them during processing and storage. In *Fish in Nutrition* (Edited by Heen E. and Kreuzer R.), pp. 86–111. Fishing News Books, London.

Mahadervan S. and Tappel A. L. (1968) Lysosomal lipase of rat liver and kidney. *J. biol. Chem.* **243**, 2849–2854.

Malbon C. C., Moreno F. J., Cabelli R. J. and Fain J. N. (1978) Fat cell adenylate cyclase and β-adrenergic receptors in altered thyroid states. *J. biol. Chem.* **253**, 671–678.

Masoro E. J. (1968) *Physiological Chemistry of Lipids in Mammals*. W. B. Saunders, Philadelphia.

McKeown B. A., Leatherland J. F. and John T. M. (1975) Effects of growth hormone and prolactin on the mobilization of free fatty acids and glucose in kokanee salmon, *Oncorhynchus nerka*. *Comp. Biochem. Physiol.* **50B**, 425–430.

McKeown B. A., John T. M. and George J. C. (1976) Effect of vasotocin on plasma GH, free fatty acids and glucose in coho salmon (*Oncorhynchus kisutch*). *Endocr. Exp.* **10**, 45–51.

Meier A. H. (1972) Temporal synergism of prolactin and adrenal steroids. *Gen. comp. Endocr.* **3**, (suppl.) 499–508.

Meisner H. and Carter J. R. (1977) Regulation of lipids in adipose tissue. *Horizons Biochem. Biophys.* **4**, 91.

Minick M. C. and Chavin W. (1970) Effect of pituitary hormone upon serum free fatty acids in goldfish (*Carassus auratus* L.). *Am. Zool.* **9**, 1082 (abstract).

Murat J. C. and Serfaty A. (1970) Au sujed d'un effect hypoglycemiant de la thyroxine chez la carpe *Cyprinus carpio* L. *C.R. Soc. Biol.* **164**, 1842–1845.

Murat J. C., Carpene C., Woo N. Y. S., Berlan M., Lafontan M. and Fraisse M. (1985) Comparative study on isolated adipocytes: specific differences in the responsiveness to hormones. In *Current Trends in Comparative Endocrinology* (Edited by Lofts B. and Holmes W. N.), Vol. 2, p. 1099. University of Hong Kong Press, Hong Kong.

Narayan K. A., Creinin H. L. and Kummerow F. Q. (1966) Disc electrophoresis of rat plasma lipoproteins. *J. Lipid Res.* **7**, 150–157.

Narayansingh T. and Eales J. G. (1975) The influence of physiological doses of thyroxine on the lipid reserves of starved and fed brook trout, *Salvelinus fontinalis* (Mitchill). *Comp. Biochem. Physiol.* **52B**, 407–412.

Nichols C. W. (1973) Somatotropic effects of prolactin and growth hormone in juvenile snapping turtle (*Chelydra serpentia*). *Gen. comp. Endocr.* **21**, 219–224.

Norris D. O. (1969) Depression of growth following radiothyroidectomy of larval Chinook salmon and steelhead trout. *Trans. Am. Fish. Soc.* **98**, 104–106.

Olley J. and Lovern J. A. (1954) The lipids of fish—5. The lipids remaining in the flesh of the haddock after extraction by acetone and ethanol–ether. *Biochem. J.* **57**, 610–619.

Pittman R. C., Khoo J. C. and Steinberg D. (1975) Cholesterol esterase in rat adipose tissue and its activation by cyclic adenosine 3':5'-monophosphate-dependent protein kinase. *J. biol. Chem.* **250**, 4505–4511.

Plisetskaya E. and Mazina T. I. (1969) The effect of hormones on NEFA content in the blood of the Baltic lamprey (*Lampetra fluviatilis* L.). *J. Evol. Biochem. Physiol.* **5**, 457.

Plisetskaya E. (1980) Fatty acid levels in the blood of cyclostomes and fish. *Environ. Biol. Fish.* **5**, 273–290.

Plisetskaya E., Woo N. Y. S. and Murat J. C. (1983) Thyroid hormones in cyclostomes and fish and their role in regulation of intermediary metabolism. *Comp. Biochem. Physiol.* **74A**, 179–187.

Rao K. S. P. and Rao K. V. R. (1984) Changes in the tissue lipid profiles of fish (*Oreochromis mossambicus*) during methyl parathion toxicity—a time-course study. *Toxicol. Lett.* **21**, 147–153.

Rich C., Biermann E. L. and Schwartz I. L. (1959) Plasma nonesterified fatty acids in hyperthyroid states. *J. clin. Invest.* **38**, 275–278.

Rizack M. A. (1961) An epinephrine-sensitive lipolytic activity in adipose tissue. *J. biol. Chem.* **236**, 657–662.

Robinson J. S. and Mead J. F. (1973) Lipid absorption and deposition in rainbow trout (*Salmo gairdnerii*). *Can. J. Biochem.* **51**, 1050–1058.

Rogie A. and Skinner E. R. (1981) Some aspects of lipid transport in the rainbow trout. *Biochem. Soc. Trans.* **9**, 59.

Rogie A. and Skinner E. R. (1985) The roles of the intestine and liver in the biosynthesis of plasma lipoproteins in the rainbow trout, *Salmo gairdnerii* Richardson. *Comp. Biochem. Physiol.* **81B**, 285–289.

Sheridan M. A. (1983) Hormonal regulation of lipid mobilization in fish: effect of epinephrine and norepinephrine on free fatty acid release from coho salmon (*Oncorhynchus kisutch*) liver incubated *in vitro*. *Am. Zool.* **23**, 1010 (abstract).

Sheridan M. A., Allen W. V. and Kerstetter T. H. (1983) Seasonal variation in the lipid composition of steelhead trout, *Salmo gairdnerii* Richardson, associated with the parr–smolt transformation. *J. Fish. Biol.* **23**, 125–134.

Sheridan M. A. and Allen W. V. (1984) Partial purification of triacylglycerol lipase isolated from steelhead trout (*Salmo gairdnerii*) adipose tissue. *Lipids* **19**, 347–352.

Sheridan M. A., Friedlander J. K. L. and Allen W. V. (1985a) Chylomicra in the serum of postprandial steelhead trout (*Salmo gairdnerii*). *Comp. Biochem. Physiol.* **81B**, 281–284.

Sheridan M. A., Woo N. Y. S. and Bern H. A. (1985b) Changes in the rates of glycogenesis, glycogenolysis, lipogenesis and lipolysis in selected tissues of the coho salmon (*Oncorhynchus kisutch*) associated with parr–smolt transformation. *J. exp. Zool.* **236**, 35–44.

Sheridan M. A. (1986) Effects of thyroxin, cortisol, growth hormone, and prolactin on lipid metabolism of coho salmon, *Oncorhynchus kisutch*, during smoltification. *Gen. comp. Endocr.* **64**, 220–238.

Sheridan M. A. and Bern H. A. (1986) Both somatostatin and the caudal neuropeptide, urotensin II, stimulate lipid mobilization from coho salmon liver incubated *in vitro*. *Reg. Pept.* **14**, 333–344.

Sheridan M. A. (1987) Effects of epinephrine and nor-

epinephrine on lipid mobilization from coho salmon liver incubated *in vitro*. *Endocrinology* **120**, 2234–2239.

Sheridan M. A., Plisetskaya E., Bern H. A. and Gorbman A. (1987) Effects of somatostatin-25 and urotensin II on lipid and carbohydrate metabolism of coho salmon, *Oncorhynchus kisutch*. *Gen. comp. Endocr.* **66**, 405–414.

Singh A. K. (1979) Effect of thyroxine and thiourea on liver and muscle energy stores in the freshwater catfish, *Heteropneustes fossilis*. *Biol. Anim. Biochem. Biophys.* **19**, 1669–1676.

Sire M. F., Lutten C. and Vernier J. M. (1981) New views on intestinal absorption of lipids in teleostean fishes: an ultrastructural and biochemical study in rainbow trout. *J. Lipid Res.* **22**, 81–94.

Sire M. F. and Vernier J. M. (1981) Etude ultrastructurale de la synthese de chylomicrons au cours de l'absorption intestinale, des lipides chez le truite. Influence de la nature des acides gras ingeres. *Biol. Cell.* **40**, 47–61.

Skinner E. R. and Rogie A. (1978) The isolation and partial characterization of the serum lipoproteins and apoproteins of the rainbow trout. *Biochem. J.* **173**, 507–520.

Skinner E. R. and Youssef A. M. (1982) The characterization of lipoprotein lipase isolated from the post-heparin plasma of the rainbow trout, *Salmo gairdnerii* Richardson. *Biochem. J.* **203**, 727.

Takashima F., Habiya T., Phan-van N. and Aid K. (1972) Endocrinological studies on lipid metabolism in rainbow trout—II. Effects of sex steroids, thyroid powder and adrenocorticotropin on plasma lipid content. *Bull. Jap. Soc. Sci. Fish.* **38**, 43–49.

Tashima L. and Cahill G. F. (1968) Effects of insulin in the toadfish *Opsanus tau*. *Gen. comp. Endocr.* **11**, 262–271.

Vaughan M. (1961) The metabolism of adipose tissue *in vitro*. *J. Lipid Res.* **2**, 293.

Vaughan M., Berger J. E. and Steinberg D. (1964) Hormone-sensitive lipase and monoglyceride lipase activities in adipose tissue. *J. biol. Chem.* **239**, 401–409.

Vaughan M. and Steinberg D. (1965) Glyceride biosynthesis, glyceride breakdown and glycogen breakdown in adipose tissue: mechanisms and regulation. In *Handbook of Physiology* (Edited by Renold A. E. and Cahill G. F.), Sect. 5, pp. 239–251. Williams & Wilkins, Baltimore, MD.

Vernier J. M. and Sire M. F. (1983) L'absorption intestinale des lipides chez la truite arc-en-ciel (*Salmo gairdnerii*). Bases biologiques de l'aquaculture, Montpellier, France 1983 IFREMER Actes de Colloques 1, 393–428.

deVlaming V. L. and Sage M. (1972) Diurnal variation in fattening response to prolactin treatment in two cyprinodontid fishes, *Cyprinodon variequatus* and *Fundulus similis*. *Contrib. Mar. Sci.* **16**, 59–63.

Walton M. J. and Cowey C. B. (1982) Aspects of intermediary metabolism in salmonid fish. *Comp. Biochem. Physiol.* **73B**, 59–80.

Watanabe T. (1982) Lipid nutrition in fish. *Comp. Biochem. Physiol.* **73B**, 3–16.